住院醫師報告
六大核心能力 (DOPS 病例應用)

外科部住院醫師 王耀鐸

105-9-3

外科部務務會議

Patient information

- Patient age / sex: 88 y/o, male
- Patient name / number: ◎ ◎ ◎ / xxxxxxx

Chief complaint

- ecchymosis over his eyelid and petechia over his limbs

Present illness

- He recept re-do TKR in the xxxx 醫院, then he was managed with celebrix for pain control
- On 8/4, he visited our OPD where ecchymosis over his eyelid and petechia over his limbs.
- INR>10, coumadin over dose was suspected

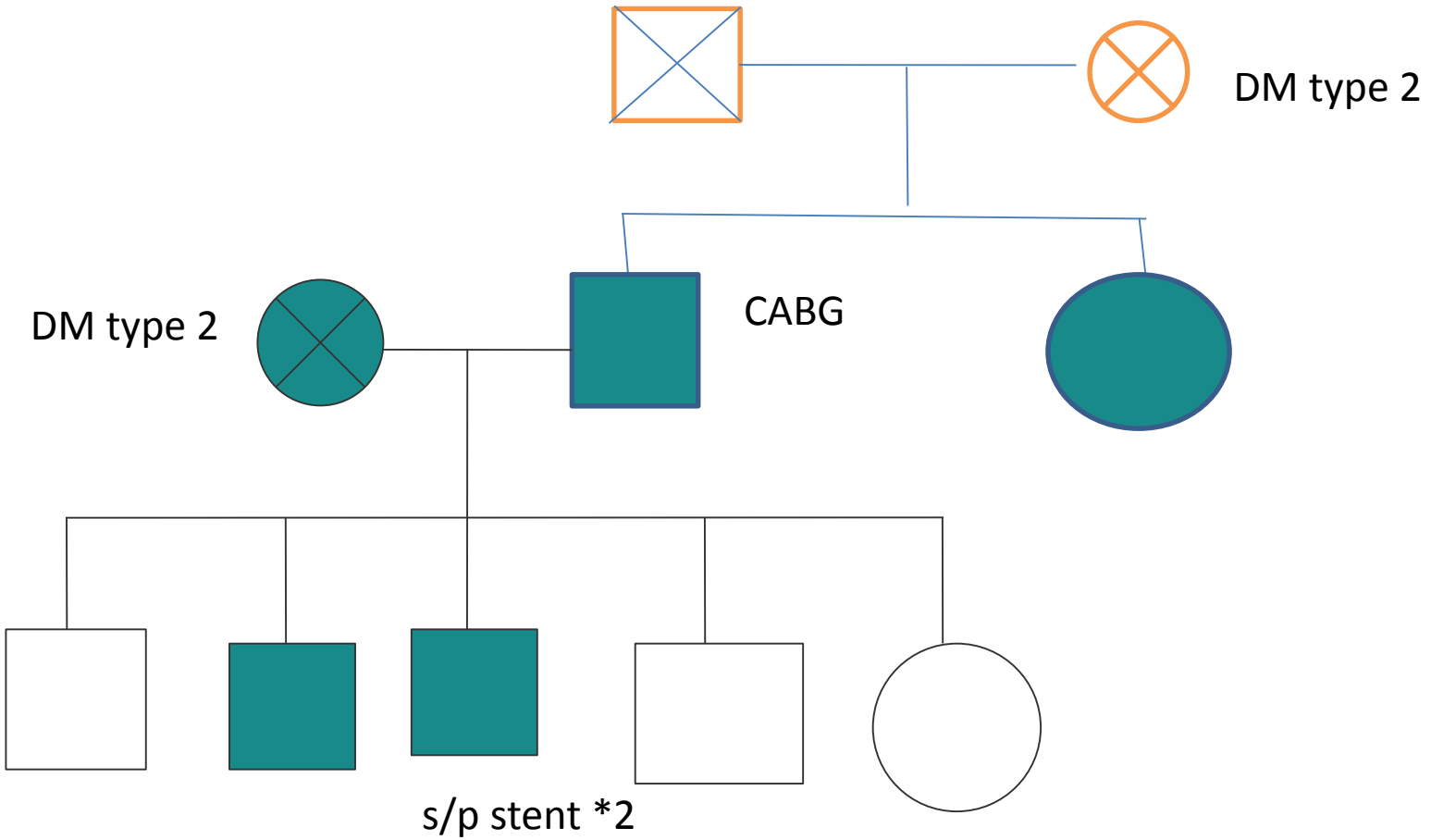
Past history & operation history

- HTN for several years
- OA knee s/p TKR *2 and under regullar medicine for pain control
- CAD with TVD and LM disease S/P CABG X4 and under regullar warfarin

Personal history

- No betel-nut, no smokimg, no alcohol
- No allerge to food and medicine

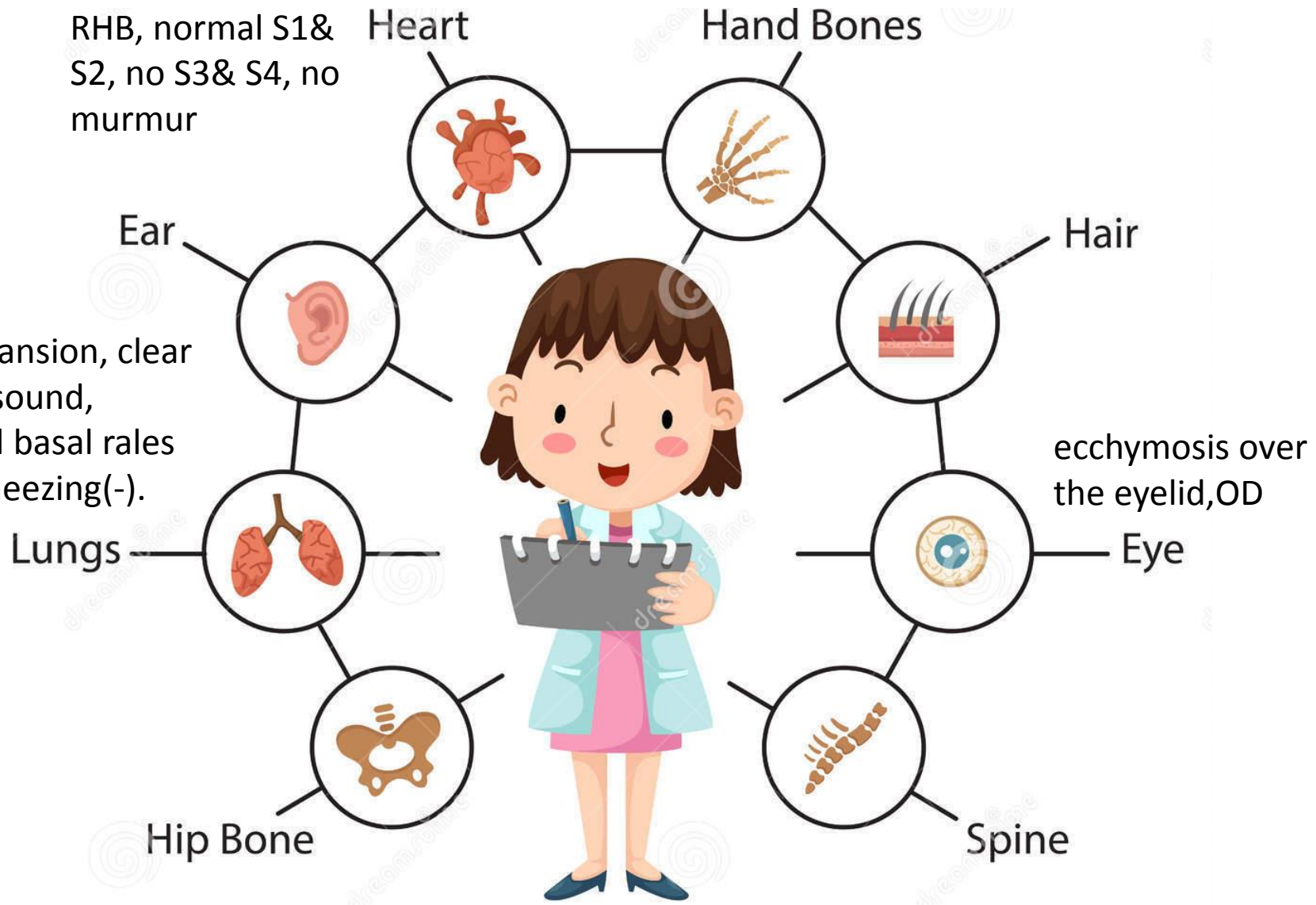
Family tree



•Review of system

- General: no malaise, no fever, no chills, no weight change
- Cardiovascular system: no chest tightness, no syncope, no palpitation
- Pulmonary system: no hemoptysis
- Gastrointestinal system: tarry stool (+), no abdomen pain or discomfort
- Neurologic system: no consciousness change, no headache

Physical examination



•Lab data

WBC	HGB	HCT	NEUT	PT
6400/uL	4.5 g/dL	13.1%	86.2 %	>100 sec
PT-INR	Ca	Mg	Na	K
> 10	8.0 mg/dL	2.09 mg/dL	135 mmol/L	5.2 mmol/L

For r/o gastric bleeding

PROCEDURE: After reviewing the risks and benefits, the patient was deemed in satisfactory condition to undergo the procedure. The heart rate, oxygen saturation, blood pressure, and response to care were monitored throughout the procedure. The physical status of the patient was re-assessed after the procedure. After obtaining informed consent, the endoscope was passed under direct vision through the mouth, and advanced to the second part of the duodenum.

FINDINGS: Esophagus: The entire esophagus was normal in appearance.
Stomach: a linear ulcer, about 1 cm, at antrum
Duodenum: The bulb and second portion including the Ampulla of Vater were normal in appearance.

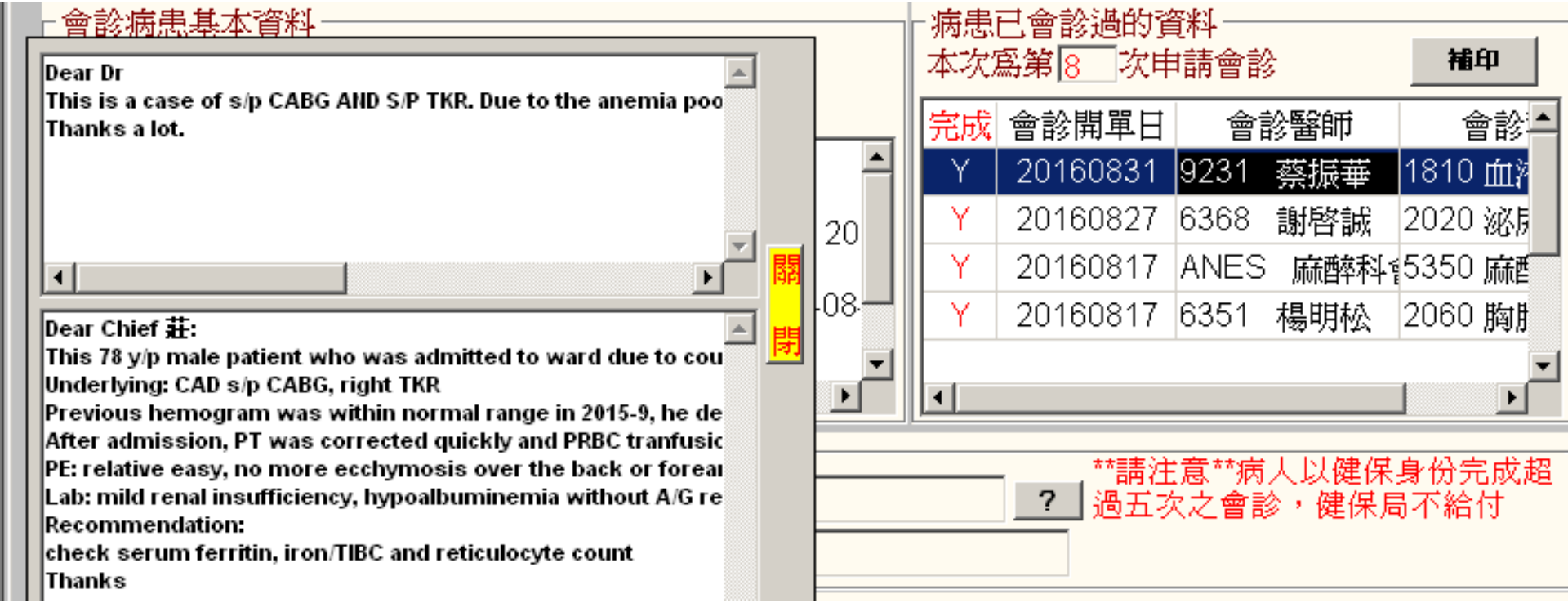
MANAGEMENT: ( ) 切片採檢 ( ) 切片摘除 ( ) 癌肉切除術
( ) 黏膜切除術 ( ) 止血
( ) 其他

IMPRESSION: 1. clean based gastric ulcer

RECOMMENDATION:
★ Explain to the patient and family members about the findings.
★ PPI 1# QDAC, on diet

COMMUNICATION:

For r/o Hema related



Diagnosis

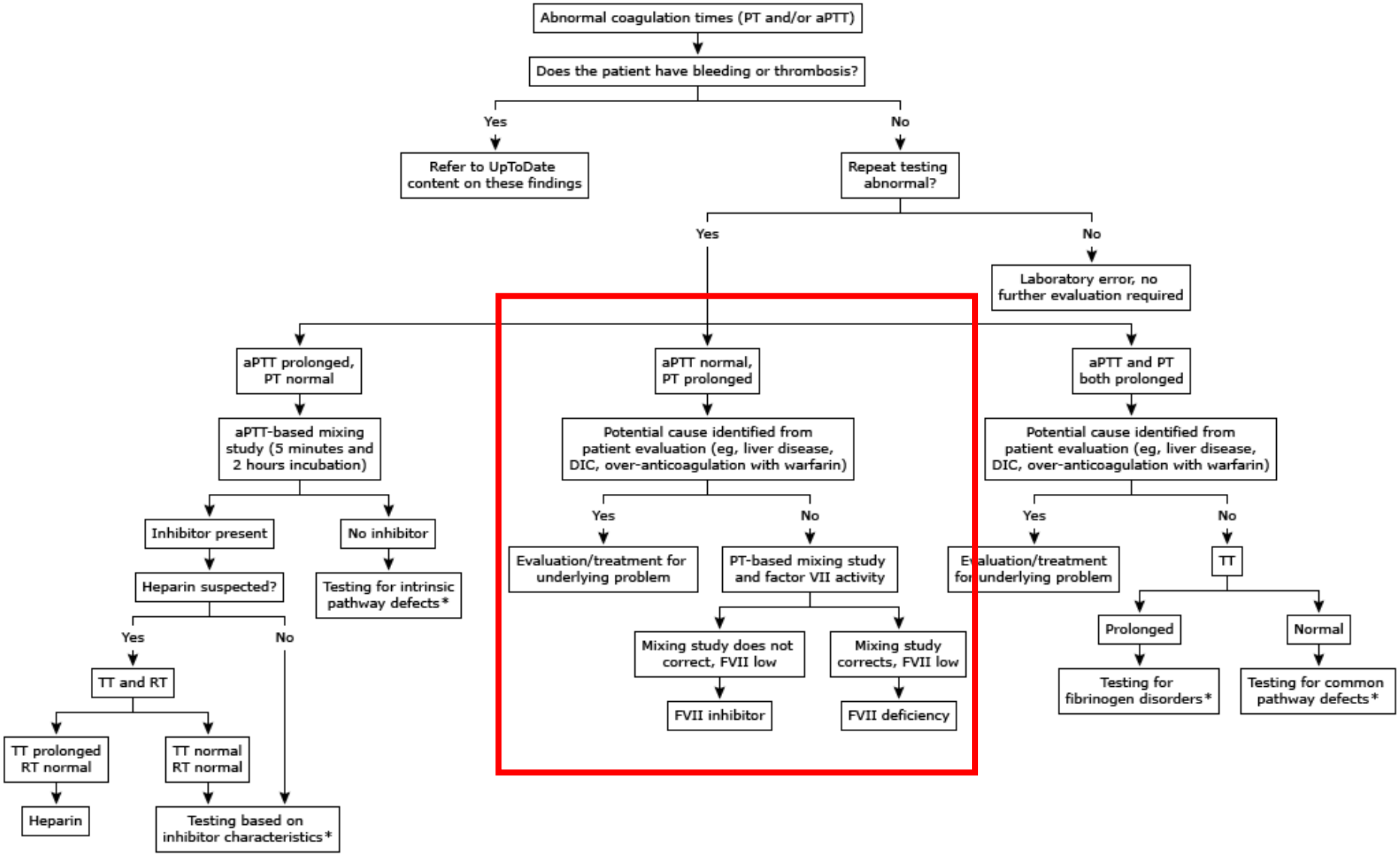
1. Coumadin over dose
2. CAD with TVD and LM disease S/P CABG X4

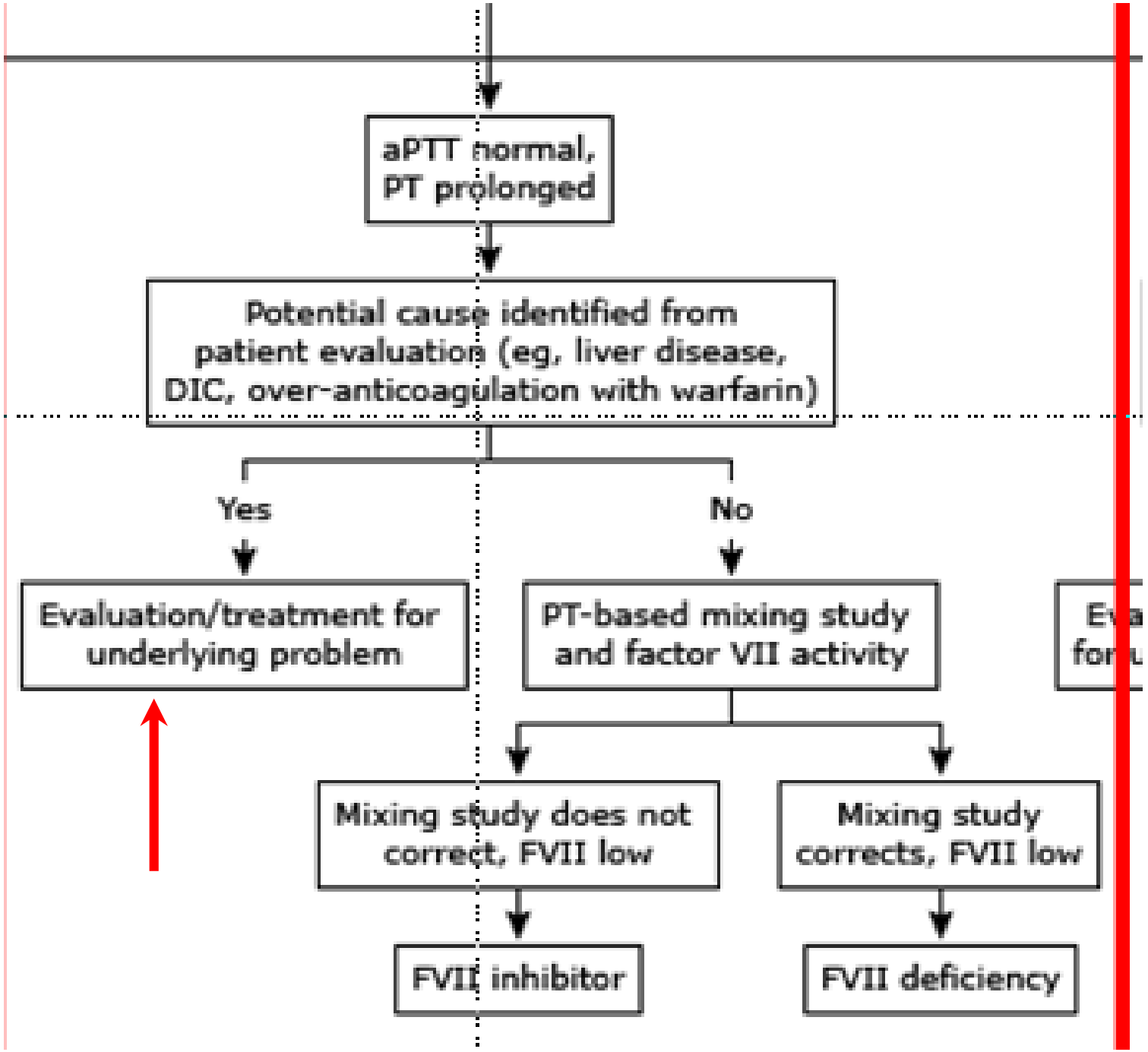
   (LIMA-CAD, SVG to RCA-D, LCx, Diag) on 2011-08-19.
3. Hypertension.
4. Anemia ,cause unknown

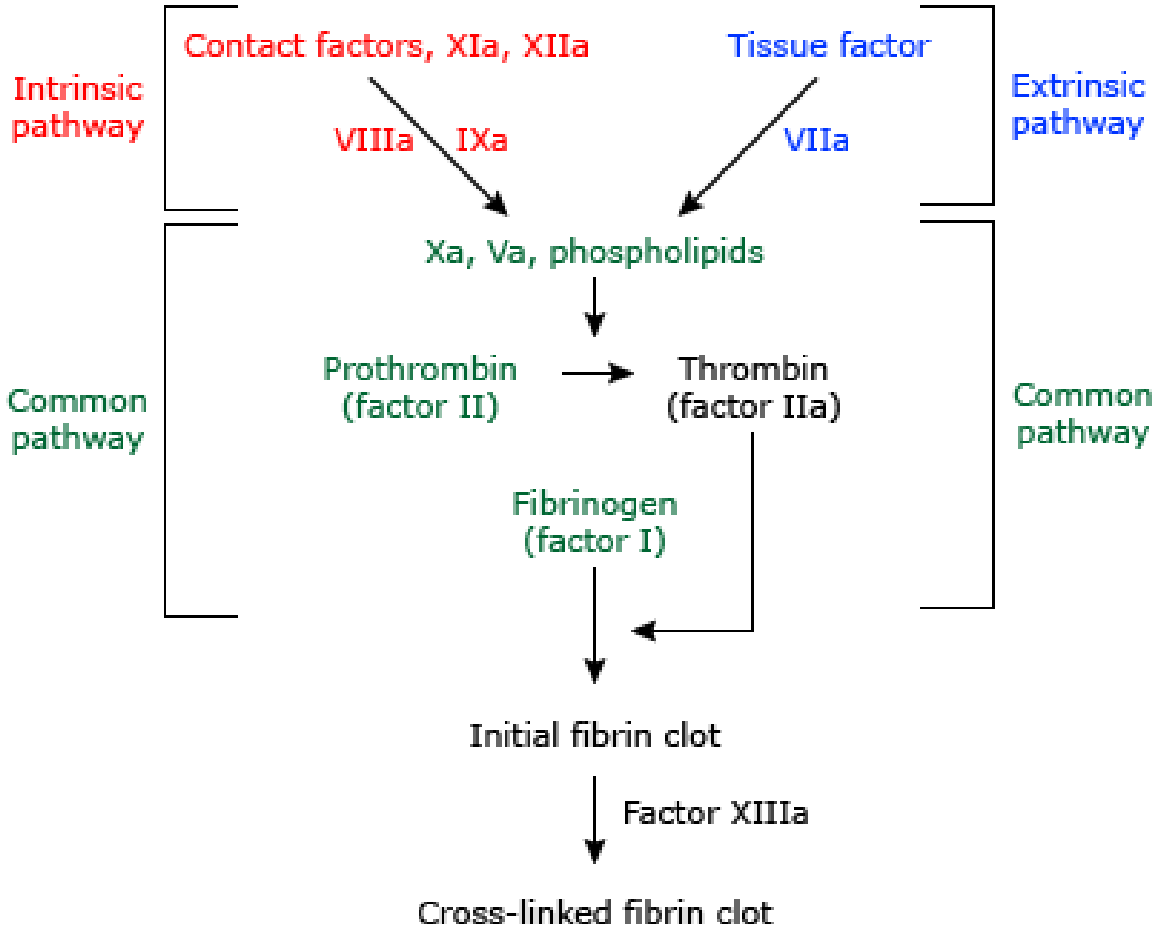
PROTHROMBIN TIME PROLONG

Introduction

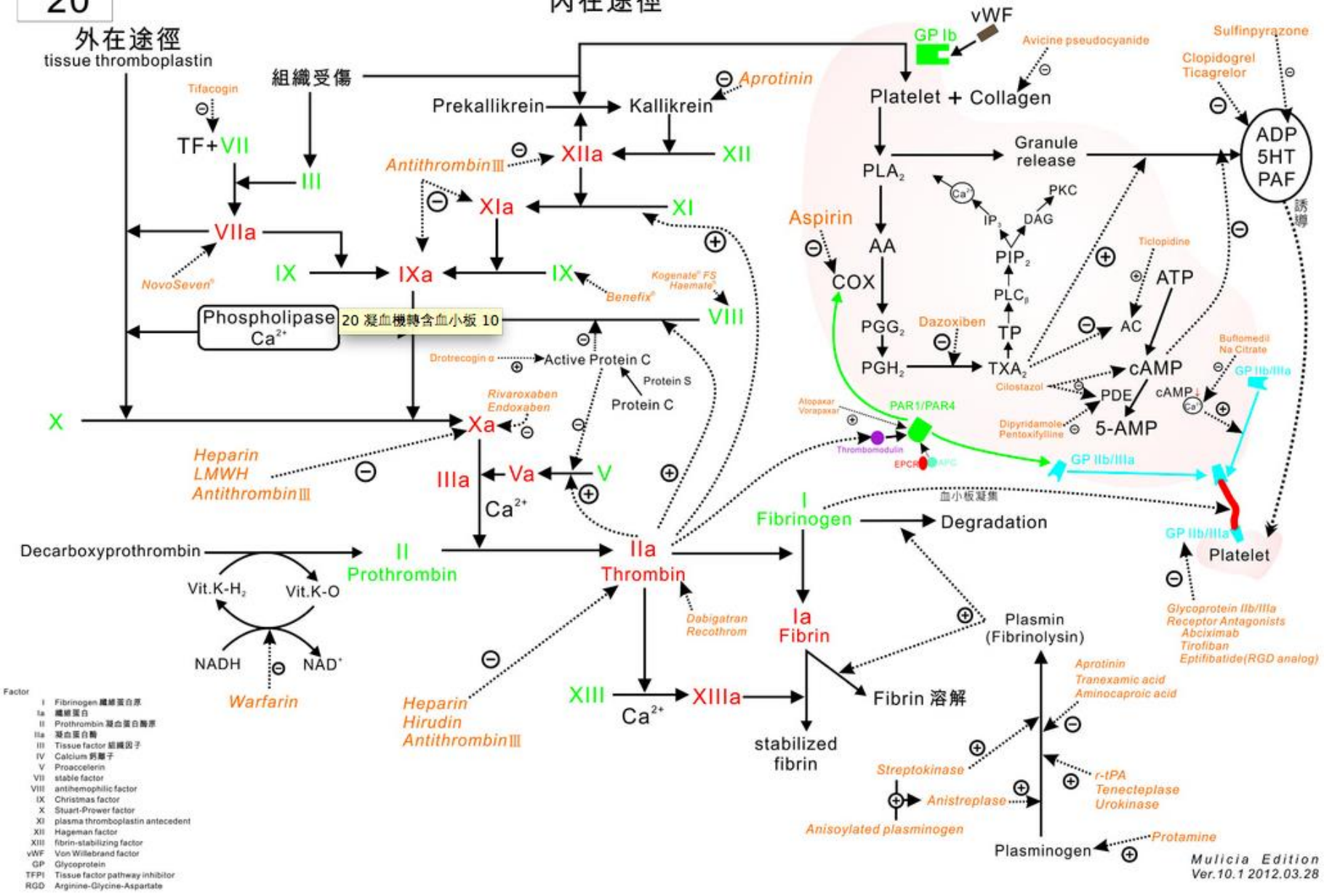
- Clotting times : the time to clot
- Ca^{2+} : coagulation factor complexed on activated cell surfaces or phospholipids.
- tissue factor for the prothrombin time [PT]
- silica or diatomaceous earth for the activated thromboplastin time [aPTT])
- PT are standardized with the international normalized ratio (INR)


Abnormal coagulation times (PT and/or aPTT)
Does the patient have bleeding or thrombosis?
Yes
No
Refer to UpToDate content on these findings
Repeat testing abnormal?
Yes
No
Laboratory error, no further evaluation required
aPTT prolonged, PT normal
aPTT normal, PT prolonged
aPTT and PT both prolonged
aPTT-based mixing study (5 minutes and 2 hours incubation)
Potential cause identified from patient evaluation (eg, liver disease, DIC, over-anticoagulation with warfarin)
Potential cause identified from patient evaluation (eg, liver disease, DIC, over-anticoagulation with warfarin)
Inhibitor present
No inhibitor
Yes
No
Yes
No
Yes
No
Heparin suspected?
Testing for intrinsic pathway defects*
Evaluation/treatment for underlying problem
PT-based mixing study and factor VII activity
Evaluation/treatment for underlying problem
TT
Yes
No
Mixing study does not correct, FVII low
Mixing study corrects, FVII low
Prolonged
Normal
TT and RT
FVII inhibitor
FVII deficiency
Testing for fibrinogen disorders*
Testing for common pathway defects*
TT prolonged RT normal
TT normal RT normal
Heparin
Testing based on inhibitor characteristics*

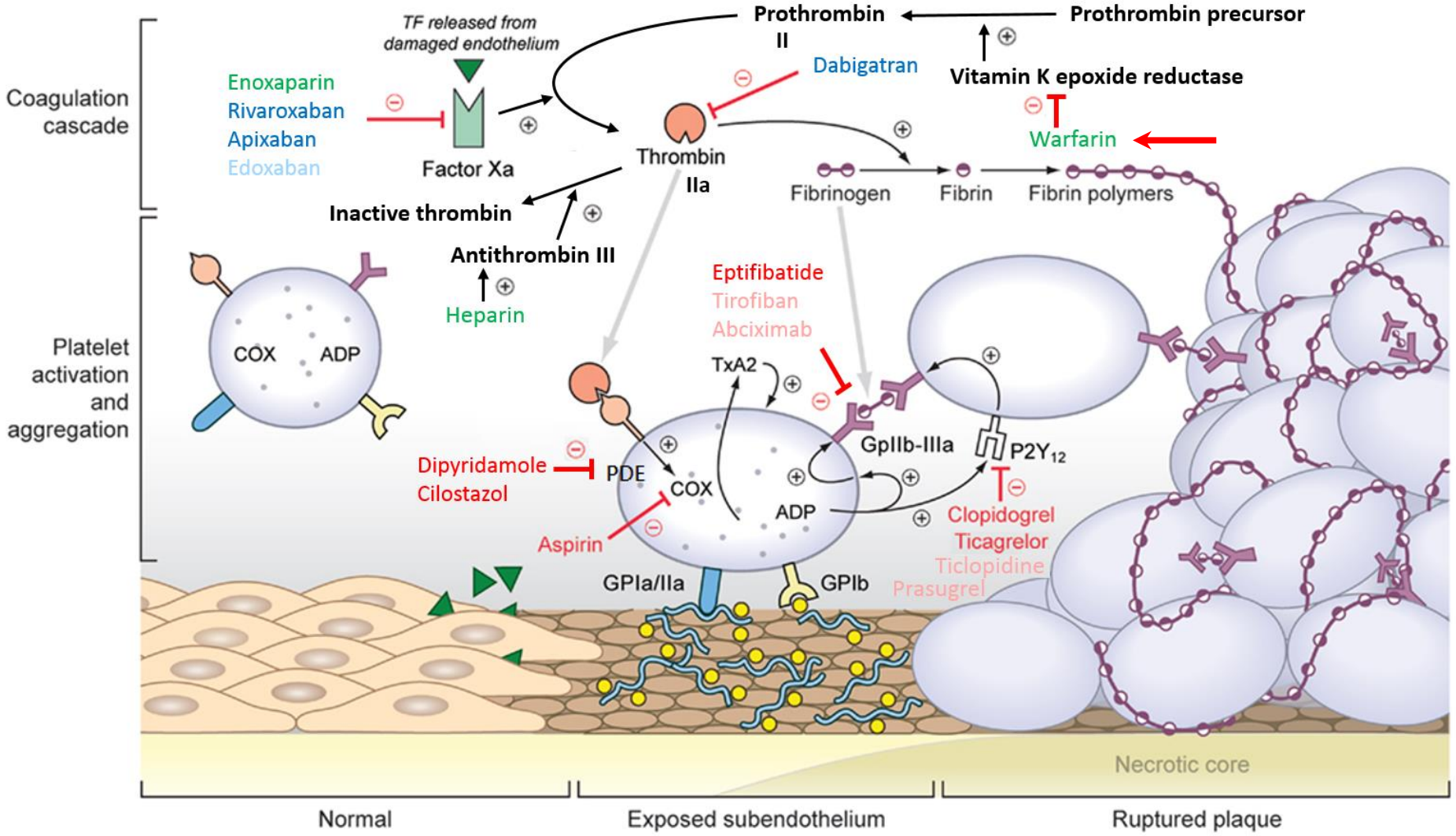

aPTT normal, PT prolonged
Potential cause identified from patient evaluation (eg, liver disease, DIC, over-anticoagulation with warfarin)
Yes
No
Evaluation/treatment for underlying problem
PT-based mixing study and factor VII activity
Mixing study does not correct, FVII low
Mixing study corrects, FVII low
FVII inhibitor
FVII deficiency



intrinsic (in red)
extrinsic (in blue)
common (in green)
Contact factors: prekallikrein and high molecular weight kininogen (HMWK).
the intrinsic (and common) : aPTT
the extrinsic (and common) : PT
thrombin time (TT)
the conversion of fibrinogen to fibrin, following the addition of exogenous thrombin. Fibrin is crosslinked through the action of factor XIII, making the final fibrin clot insoluble in 5 Molar urea or monochloroacetic acid.


內在途徑
外在途徑
tissue thromboplastin
組織受傷
Tifacogin
TF+VII
III
VIIa
NovoSeven®
IX
IXa
IX
Kogenate® FS Haemate®
Benefix®
VIII
Phospholipase Ca2+
20 凝血機轉含血小板 10
Drotrecogin α
Active Protein C
Protein S
Protein C
X
Xa
Rivaroxaben Endoxaban
Heparin LMWH Antithrombin III
IIIa
Va
V
Ca2+
Decarboxyprothrombin
II Prothrombin
Vit.K-H2
Vit.K-O
NADH
NAD+
Warfarin
IIa Thrombin
Dabigatran Recothrom
Heparin Hirudin Antithrombin III
XIII
Ca2+
XIIIa
Prekallikrein
Kallikrein
Aprotinin
Antithrombin III
XIIa
XII
XIa
XI
I Fibrinogen
Degradation
Ia Fibrin
stabilized fibrin
Fibrin 溶解
Plasmin (Fibrinolysin)
Aprotinin
Tranexamic acid
Aminocaproic acid
Streptokinase
Anistreplase
Anisoylated plasminogen
r-tPA
Tenecteplase
Urokinase
Plasminogen
Protamine
vWF
GP Ib
Avicine pseudocyanide
Platelet + Collagen
Sulfinpyrazone
Clopidogrel
Ticagrelor
ADP
5HT
PAF
誘導
Granule release
PLA2
Aspirin
AA
COX
PGG2
PGH2
TXA2
Dazoxiben
Ca2+
IP3
DAG
PKC
PIP2
PLCβ
TP
Ticlopidine
ATP
AC
cAMP
Cilostazol
PDE
5-AMP
cAMP↓
Ca2+
Buflomedil Na Citrate
GP IIb/IIIa
Alopaxar Vorapaxar
PAR1/PAR4
Thrombomodulin
EPCR
APC
Dipyridamole Pentoxifylline
GP IIb/IIIa
血小板凝集
GP IIb/IIIa
Platelet
Glycoprotein IIb/IIIa Receptor Antagonists
Abciximab
Tirofiban
Eptifibatide(RGD analog)
Factor
I Fibrinogen 纖維蛋白原
Ia 纖維蛋白
II Prothrombin 凝血蛋白酶原
IIa 凝血蛋白酶
III Tissue factor 組織因子
IV Calcium 鈣離子
V Proaccelerin
VII stable factor
VIII antihemophilic factor
IX Christmas factor
X Stuart-Prower factor
XI plasma thromboplastin antecedent
XII Hageman factor
XIII fibrin-stabilizing factor
vWF Von Willebrand factor
GP Glycoprotein
TFPI Tissue factor pathway inhibitor
RGD Arginine-Glycine-Aspartate
Mulicia Edition
Ver.10.1 2012.03.28

抗血小板藥物 & 抗凝血藥物作用機轉



Ref: Front. Pharmacol. 24 October 2011, Figure 1

Bleeding symptoms	Bleeding disorder	
	Platelet defects (qualitative or quantitative)	Clotting factor deficiencies (eg, factor VIII or factor IX deficiencies)
Overview of bleeding events	**Mucocutaneous bleeding (oral cavity, nasal, gastrointestinal, and genitourinary sites)**	**Deep tissue bleeding (including joints and muscles)**
Excessive bleeding after minor cuts	Yes	Not usually
Petechiae	Common	Uncommon
Ecchymoses	Generally small and superficial; may be significant, depending upon the defect or degree of thrombocytopenia	May develop large subcutaneous and soft tissue hematomas
Hemarthroses, muscle hematomas	Uncommon	Common in severe deficiency states or in association with injury in those with mild to moderate deficiency states
Bleeding with invasive procedures, including surgery	Often immediate, with degree of bleeding dependent upon the severity of the defect, ranging from none (eg, mild degrees of thrombocytopenia or mild platelet function defect) to mild to severe (eg, Glanzmann thrombasthenia)	May be associated either with procedural bleeding or delayed bleeding, depending upon the type and severity of the defect

Clinical circumstance(s)	PT testing alone	aPTT testing alone	PT and aPTT testing combined
Monitoring anticoagulation			
Warfarin only (without heparin)	√	–	–
Therapeutic unfractionated heparin (without warfarin)	–	√	–
Transitions between heparin and warfarin therapy	–	–	√
Patient assessment			
Assessment of patients with signs or symptoms of hemorrhage or thrombosis	–	–	√
Assessment of patients with history of condition known to be associated with risk of bleeding or thrombosis due to **extrinsic** coagulation pathway abnormalities, either genetic or acquired	√	–	–
Assessment of patients with history of condition known to be associated with risk of bleeding or thrombosis due to **intrinsic** coagulation pathway abnormalities, either genetic or acquired	–	√	–
Assessment of risk of hemorrhage or thrombosis in patients who are going to have a medical intervention known to be associated with increased risk of bleeding or thrombosis	–	–	√

Clinical uses of the PT/INR

- Evaluation of unexplained bleeding
- Diagnosing disseminated intravascular coagulation
- Obtaining a baseline value prior to initiating anticoagulation
- Monitoring warfarin therapy
- Assessment of liver synthetic function

Causes of prolonged PT

- Vitamin K antagonists – warfarin interfere with post-translational modifications of procoagulant factors II, VII, IX, and X
- Other anticoagulants – Heparins and fondaparinux inhibit thrombin and/or factor Xa ; PT may become elevated at heparin concentrations above 1 unit/mL ; All of the available direct acting anticoagulants prolong the PT, including argatroban, dabigatran, rivaroxaban, apixaban, and edoxaban.

- Vitamin K deficiency – impaired nutrition, prolonged use of broad spectrum antibiotics, or fat malabsorption syndromes ; if severe , both the PT and aPTT may be prolonged
- Liver disease – decreased production of both vitamin K-dependent and vitamin K-independent clotting factors ; predominant effect on factor VII

- DIC – coagulation factors become consumed and depleted ; result in prolonged PT and aPTT.
- Factor deficiency – extrinsic pathway ; includes deficiency of fibrinogen and factors II, V, VII, or X
- Antiphospholipid antibodies – Lupus anticoagulants with specificity for prothrombin may cause hypoprothrombinemia and prolongation of the PT

PT	aPTT	Causes of test result pattern
Prolonged	Normal	**Inherited**
		Factor VII deficiency
		Acquired
		Mild vitamin K deficiency
		Liver disease
		Warfarin administration*
		Acquired inhibitor of factor VII
		Lupus anticoagulant (more commonly causes isolated prolonged aPTT; may be associated with thrombosis rather than bleeding)

Normal	Prolonged	**Inherited**
		Deficiency of factors VIII, IX, or XI
		Deficiency of factor XII, prekallikrein, or HMW kininogen (not associated with a bleeding diathesis)
		von Willebrand disease (variable)
		Acquired
		Heparin administration*
		Inhibitor of factors VIII, IX, XI, or XII
		Acquired von Willebrand disease
		Lupus anticoagulant (may be associated with thrombosis rather than bleeding)

Prolonged	Prolonged	**Inherited**
		Deficiency of prothrombin, fibrinogen, or factors V or X
		Combined factor deficiencies
		Acquired
		Liver disease
		Disseminated intravascular coagulation
		Supratherapeutic doses of anticoagulants
		Severe vitamin K deficiency
		Combined heparin and warfarin administration
		Direct thrombin inhibitor administration (eg, argatroban, dabigatran)*
		Direct factor Xa inhibitor administration (eg, rivaroxaban, apixaban, edoxaban)
		Fondaparinux administration (slight prolongation)
		Inhibitor of prothrombin, fibrinogen, or factors V or X
		Primary amyloidosis-associated factor X deficiency
		Anticoagulant rodenticide poisoning

Drug class	Drug	Brand name(s)	PT	aPTT	Anti-factor Xa activity
Vitamin K antagonists	Warfarin	Coumadin, Jantoven	↑	↑/–*	–
	Acenocoumarol	Sintrom	↑	↑/–*	–
Heparins	Unfractionated heparin		–¶	↑	↑
	LMW heparins Enoxaparin Dalteparin Nadroparin	 Lovenox Fragmin Fraxiparine	–	↑/–	↑
	Fondaparinux	Arixtra	–	↑/–	↑
Direct thrombin inhibitors	Argatroban	Acova	↑	↑	–
	Dabigatran	Pradaxa	↑/–	↑	–
Direct factor Xa inhibitors	Rivaroxaban	Xarelto	↑/–	↑/–	↑/–Δ
	Apixaban	Eliquis	↑/–	↑/–	↑/–Δ

Disorder	Plt	PT	aPTT	TT	Fib
Vasculopathies, connective tissue diseases, or collagen disorders affecting skin	Normal	Normal	Normal	Normal	Normal or increased*
Thrombocytopenia	Low	Normal	Normal	Normal	Normal
Qualitative platelet abnormalities	Normal or low ¶	Normal	Normal	Normal	Normal
Hemophilia A (factor VIII deficiency)	Normal	Normal	Long	Normal	Normal
von Willebrand disease	Normal Δ	Normal	Long ◇	Normal	Normal
Disseminated intravascular coagulation	Low	Long	Long	Long	Low

May increase INR
Acetaminophen
Allopurinol
Amiodarone
Androgens (eg, methyltestosterone, oxandrolone, testosterone)
Antibiotics
▪ Penicillins (eg, amoxicillin, amoxicillin-clavulanate) Exceptions: Dicloxacillin and nafcillin may decrease the INR
▪ Doxycycline
▪ Cephalosporins
▪ Fluoroquinolones (eg, ciprofloxacin, levofloxacin, moxifloxacin, norfloxacin)
▪ Macrolides (eg, azithromycin, erythromycin, clarithromycin)
▪ Metronidazole
▪ Trimethoprim-sulfamethoxazole

Azole antifungals (eg, fluconazole, miconazole [oral], voriconazole)*
Cancer therapies
▪ Capecitabine
▪ Fluorouracil (5-FU)
▪ Imatinib
▪ Tamoxifen
Cholesterol-lowering agents (eg, gemfibrozil, fenofibrate, fluvastatin, lovastatin, rosuvastatin, simvastatin) Exception: Cholestyramine may decrease the INR
Cimetidine
Glucocorticoids (eg, prednisone, methylprednisolone)
Omeprazole (case reports with other proton pump inhibitors)
Serotonin reuptake inhibitors (eg, duloxetine, fluoxetine, fluvoxamine, sertraline, venlafaxine)
Sitaxentan (not available in United States)
Tramadol

May decrease INR
Antibiotics
▪ Dicloxacillin
▪ Griseofulvin
▪ Nafcillin
▪ Rifampin
Azathioprine
Enzyme-inducing antiepileptic drugs (eg, carbamazepine, phenobarbital, phenytoin [mixed effects described])
Cholestyramine
Herbal remedies (eg, St John's wort)*
Ritonavir
Sucralfate
Vitamin K

食品與藥品(warfarin)可能之交互作用

可能之交互作用	降低抗凝血作用	加強抗凝血作用
食品-食材名稱	輔酶 Q10 貫葉連翹 (St. John's wort) 維生素C (高劑量) 維生素K 人參# 綠茶* 豆奶 (大豆)*	洋蔥 蔓越莓 當歸 辣椒 葡萄柚 魚油 大蒜 鳳梨酵素 丹參 銀杏 薑 芒果 人參# 維生素E (>400 IU/天) 甲殼素* 葡萄子*

資料來源：

1.藥品仿單

2.Paula G. Am Fam Physician. 2008;77(1):73-78.

3.Anne M. Holbrook. Arch Intern Med. 2005;165:1095-1106.

4.Nadine A. British Journal of Haematology. 2005;130:777-780.

#文獻顯示有增強或降低抗凝血作用。

*文獻來源:僅少數案例報告或動物實驗佐證。

全國健康食品及膠囊錠狀食品非預期反應通報系統 http://hf.doh.gov.tw

行政院衛生署 關心您

含維他命K的食物表

食物	含大量維他命K	含中量維他命K	含少量維他命K
可進食量	避免進食	按營養師建議進食合適份數	固定及適量進食
蔬菜類	芥蘭、莧菜、菠菜、通菜、韮菜	西蘭花、芥菜、西生菜、椰菜、茼蒿、唐生菜、西洋菜，小唐菜、青蘿蔔、菜心、椰菜花、絲瓜	其他蔬菜如：節瓜、粉葛、蘆筍、茄子、紹菜、粟米、蕃茄、韮王、苦瓜、蓮藕、薯仔、慈菇、芋頭、白蘿蔔、紅蘿蔔
水果	棗類（如紅棗）		果汁、水果（牛油果除外）
豆類		黃豆、扁豆、綠豆、三角豆	豆腐
五穀			飯、粉、麵、麵包、餅乾
肉類			豬、牛、羊，家禽、魚、蛋
奶類			牛奶及牛奶製品
其他	紫菜、綠茶（如龍井、碧螺春、日本綠茶）		調味品（如鹽、糖、豉油、橄欖油、花生油、粟米油）

*以上資料由確進糖尿專科中心高級註冊營養師林思為小姐提供。

新舊口服抗凝血藥之藥理學比較

學名	Dabigatran	Rivaroxaban	Apixaban	Warfarin
作用標的	Thrombin	Factor Xa	Factor Xa	Vitamin K dependent coagulation factor
給藥間隔	每日兩次	每日一次	每日兩次	每日一次
生體可用率	6%	66-100%	>50%	>95%
藥物達尖峰時間	0.5-2小時	2-4小時	1-4小時	90分
半衰期	12-14小時	9小時	8-13小時	36-42小時
腎臟排除	80%	66%	25%	92%
蛋白結合	35%	90%	90%	99%
藥物交互作用	P-gp 抑制劑和誘導劑	合併P-gp和CYP3A4抑制劑和誘導劑	合併P-gp和CYP3A4抑制劑和誘導劑	CYP2C9, 1A2, 3A4
解毒劑	無(建議洗腎)	無(建議PCCs)	無(建議PCCs)	PCCs或FFP或vitamin K

縮寫：FFP-fresh frozen plasma, PCCs-prothrombin complex concentrates, P-gp-P-glycoprotein

Warfarin V.S. Dabigatran

項目	Warfarin[1]	Dabigatran[2]
作用機轉	維他命k拮抗劑，抑制凝血因子II、VII、IX & X的活性，間接產生抗凝血的效果。	直接抑制凝血酶結合，產生抗凝血的效果。
定期血液監測	需監測凝血時間(INR)，以避免藥物劑量過量發生出血或劑量不足而導致中風。	不需要
療效	Warfarin 對於心房顫動患者可使中風相對危險性降低64%，但同時也夾帶顱內出血的高風險。	**較Warfarin能再進一步減少35%的中風或全身性栓塞風險**，效果顯著；且降低59-70%顱內出血的機率，大大提升安全性。
出血風險	高	較低
藥物交互作用	與眾多中西藥物交互作用，包括綜合維他命、銀杏等	與其他藥物交互作用機率低
食物交互作用	如豬肝、綠色蔬菜(含維他命K)如菠菜、高麗菜、魚油、芒果、洋蔥、大蒜、海帶等等	不需要改變飲食習慣
副作用	倦怠、頭痛、皮疹、癢疹、食慾不振、噁心、嘔吐、腹痛、腹瀉、腸胃出血、血尿、白血球減少、肝損傷、黃疸、骨質疏鬆、咳血	消化不良
健保給付	有	無

[1] Graeme J. Hankey and John W. Eikelboom, Dabigatran Etexilate: A New Oral Thrombin Inhibitor, Circulation 2011;123;1426-1450

[2] Hart RG, Benavent O, McBride R, Pearce LA. Antithrombotic therapy to prevent stroke in patients with atrial fibrillation: a meta-analysis. Ann Intern Med 1999; 131(7): 492-501.

手術前停用抗凝血、抗血小板藥物時間

Med.Data SPEAK

藥物		抗凝血藥物						抗血小板藥物			
		Rivaroxaban		Dabigatran		Apixaban		Warfarin	Aspirin	Clopidogrel	Ticagrelor	Ticlopidine
院內品項		Xarelto 拜瑞妥		Pradaxa 普栓達		Eliquis 艾必克凝		5 天	7-10 天	Plavix 保栓通	Brilinta 百無凝	10-14 天
腎功能	出血風險	低	高	低	高	低	高	INR ≤ 1.4		5 天	5 天	
CrCl>80 ml/min		≥ 1 天	≥ 2 天	≥ 1 天	≥ 2 天	≥ 1 天	≥ 2 天					
CrCl 50-80 ml/min				≥ 1.5 天	≥ 3 天							
CrCl 30-50 ml/min				≥ 2 天	≥ 4 天							
CrCl 15-30 ml/min		≥ 1.5 天		未核准使用		≥ 1.5 天						
CrCl <15 ml/min		未核准使用										

CrCl: Creatinine Clearance

Ref: EHRA practical guide for use of the new oral anticoagulants 2013, ACCP perioperative management of antithrombotic therapy 2012, UpToDate

Wafarin 中毒時之處理

INR	是否出血?	UpToDate建議處置	與ACCP 2012不同處
<5	無	1. 降低warfarin dose或停幾個dose 2. 密切監測INR，以調整劑量。	
5-9	無	1. 停一個dose，給口服低劑量(1-2.5 mg)的vit K。 2. 密切監測INR，等INR正常再重新給warfarin並調整劑量。	INR 4.5-10，不需常規給vit K，除非是高風險出血的病人。
>9	無	1. 停用warfarin，給口服低劑量(2.5-5 mg)的vit K。 2. 密切監測INR，等INR正常再重新給warfarin並調整劑量。	
不管INR 嚴重出血甚至有生命危險		1. 停用warfarin，給slow IV infusion (10 mg)的vit K，FFP或prothrombin complex concentration。 2. 密切監測INR，等INR正常再重新給warfarin並調整劑量。	

Warfarin	Amiodarone	口服抗凝血藥的低凝血酶原反應經由併用 Amiodarone 而增強	Amiodarone 抑制 R- and S-enantiomers of Warfarin 經由 CYP1A2, CYP2C9 的代謝	併用 Amiodarone 時的最初6-8周須密切監測 INR。若 Amiodarone 的維持劑量分別為 100, 200, 300, or 400 mg/day，則須減少 Warfarin 劑量大約 25%, 30%, 35%, or 40%，但通常須減少 30-50% Warfarin 的劑量。停用 Amiodarone 後，此影響至少持續 1.5-4 個月，所以需要不斷調整 Warfarin 劑量
Warfarin	Azole Antifungals： Fluconazole Miconazole Voriconazole	增加 warfarin 的濃度與出血的風險	抑制 warfarin 的 CYP2C9 代謝	若併用或停止併用 AZOLE ANTIFUNGAL AGENT ，至少須每兩天監測 PT 和 INR 值，必要時調整 Warfarin 劑量
Warfarin	Barbiturates： Phenobarbital	BARBITURATES 減少抗凝血劑的作用	藉由誘發肝微粒體酵素來增加抗凝血劑代謝清除率	病人併用 BARBITURATES 也須調整抗凝血劑的劑量。終止 BARBITURATE 的治療也須減少抗凝血劑的劑量，並監測病人幾週。建議使用 benzodiazepine.
Warfarin	Metronidazole	Warfarin 的抗凝血作用增加，亦會增加出血的風險	METRONIDAZOLE 會減少 Warfarin 的代謝	若須並用，須嚴密監測病人出血的症狀。建議使用低劑量 Warfarin
Warfarin	Quinolones 類： Levofloxacin Moxifloxacin	Warfarin 的抗凝血作用增加	Unknown.	建議選用非 QUINOLONE 類抗生素。當並用或停止使用 QUINOLONES 時，須適時調整 Warfarin 劑量，並經常性監測抗凝血劑活性

Warfarin	Macrolide Antibiotic 類： Azithromycin Clarithromycin Erythromycin	Warfarin 的抗凝血作用增加，亦會增加出血的風險	Warfarin 的身體清除率降低	若並用或停止並用 Macrolide 類抗生素幾天後，須經常性監測抗凝血劑作用和適時調整劑量
Warfarin	Tetracyclines： Minocycline Tetracycline	Warfarin 的抗凝血作用增加	TETRACYCLINES 會直接影響止血(hemostasis)	若要並用，則須經常性監測抗凝血劑作用和適時調整 Warfarin 劑量。指導病人有關出血的前兆和症狀
Warfarin	Fibric Acids： Gemfibrozil Fenofibrate	warfarin 的濃度不受影響，但 Fibric Acids 會增加口服抗凝血劑的低凝血酶原反應，而造成出血和死亡	凝血因子合成受影響	若須並用則須嚴密觀察 INR 值，必要時調整抗凝血劑劑量，建議病人及時反應不正常出血或瘀青
Warfarin	Salicylates： Aspirin	Warfarin 的抗凝血作用增加，增加 Aspirin 造成胃黏膜出血和血小板功能異常的機率	Complicated.	若要並用，則須經常性監測抗凝血劑作用和適時調整 Warfarin 劑量，指導病人及時反應不正常出血或瘀青
Warfarin	NSAIDs： Ibuprofen KeTorolac Diclofenac	增加抗凝血劑作用和出血的風險	胃腸道刺激和血小板功能低下	嚴密監控病人，並指導病人及照顧者出血的相關症狀
Warfarin	COX-2 Selective NSAID： Celecoxib	Warfarin 的抗凝血作用增加	Unknown.	當 NSAID 開始或停止或改變劑量時，並用抗凝血劑濃度須嚴密監控
Warfarin	HMG-CoA Reductase Inhibitor： Fluvastatin	Warfarin 的抗凝血作用增加	抑制 warfarin 的 CYP2C9 和 CYP3A4 代謝，進而減少 S- and R- Warfarin 的	若並用或停止並用，須經常性監測抗凝血劑作用。Atorvastatin and pravastatin 和 Warfarin 不會有交互作用.

	Lovastatin Rosuvastatin Simvastatin		清除率	
Warfarin	Thyroid Hormones	增加出血的風險	在利用 thyroid hormone 治療中，vitamin K 依賴性凝血因子被加速排出已有相關文獻	嚴密觀察臨床出血症狀和監測凝血指標。若並用，須降低口服抗凝血劑的劑量。相反的，若終止 THYROID HORMONE 治療，則須增加抗凝血劑的劑量
Warfarin	Thioamines： Methimazole, Propylthiouracil	抗凝血劑和 THIOAMINES 的併用治療會改變口服抗凝血劑的作用.	Unknown.	嚴密觀察臨床出血症狀和監測凝血指標。必要時調整口服抗凝血劑劑量
Warfarin	Antineoplastic Agent： Carboplatin, Cyclophosphamid, Etoposide, Fluorouracil, Gemcitabine, Paclitaxel	Warfarin 的抗凝血作用增加	有可能與蛋白置換有關，抑制 Warfarin 代謝或抑制凝血因子的合成	化療期間小心監測凝血指標，必要時調整 Warfarin 劑量
Warfarin	Sulfinpyrazone	Warfarin 的抗凝血作用增加，也增加出血的風險	SULFINPYRAZONE 的代謝物使 Warfarin 的代謝減慢	嚴密監測凝血指標，並用治療初期須減少 Warfarin 劑量，當停止並用時可增加 Warfarin N 劑量
Warfarin	Sulfonamides	Warfarin 的抗凝血作用增加，也增加出血的風險	尚未明瞭，但 TRIMETHOPRIM/SULFAMETHOXAZOLE (TMP-SMZ) 會抑制 S-warfarin 的肝代謝	監測抗凝血劑 Warfarin 作用，必要時調整 Warfarin 劑量

Warfarin	Tamoxifen	口服抗凝血劑的抗凝血酶原反應作用增強，亦會增加出血的可能性	Unknown.	若須並用，則下降低口服抗凝血劑的劑量，並持續監測 PT 和調整劑量
Warfarin	Androgens(17-alkyl)：Danazol	17-ALKYL ANDROGENS 可使口服抗凝血劑的抗凝血酶原反應作用增強	Unknown	盡可能避免使用，若須並用，則須減少口服抗凝血劑的劑量，並監測抗凝血劑作用和指標(PT)
Warfarin	Vitamin E	抗凝血作用增加	VITAMIN E 會干擾 vitamin K 依賴性凝血因子，因此增加口服抗凝血劑的反應	須嚴密觀察口服抗凝血劑的低凝血酶原反應，並監測凝血指標，適時降低抗凝血劑劑量
Tissue plasminogen activator	Warfarin	增加嚴重出血的作用	加成或協同的作用	並用是禁忌的

資料來源：

Drug interaction facts 2011 :the authority on drug interactions /[editor] David S. Tatro.

http://www.crediblemeds.org/healthcare-providers/drug-drug-interaction

Hospital course

2016/08/04~2016/08/10

Hold plavix

2016/08/07

PT INR : 1.32 → add Plavix 1# po QD

住院醫師六大核心能力

病人照護(Patient Care)

醫學知識(Medical Knowledge)

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制度下之臨床工作(Systems-based practice)